TRANSIENT GLOBAL AMNESIA – A CLINICAL STUDY

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ABSTRACT

Introduction: Transient global amnesia (TGA) is defined as a selective deficit of the memory that appears suddenly and lasts less than 24 hours. The precise pathophysiology of transient global amnesia is not clear; various brain regions may possibly be affected

Patients and method: Our series of patients with TGA were included in a prospective study during 2004-2009. The diagnosis was based on positive clinical signs and negative symptoms for other neurological disease. We recorded: risk factors, precipitating events, associated symptoms, duration, incidence in daytime and season, recurrence. History of toxic substance intake was exclusion criteria. Laboratory tests, EEG, ECG and brain MRI were performed.

Results: 18 patients were diagnosed as TGA, 6 (33.33%) men, 12 (66.66%) women, age: 36-72-year-old. 2 (11.11%) had no risk factors. We found personality changes in 12 (66%), high blood pressure in 6 (33%), history of migraine in 5 (27.77%), previous stroke in 3 (16.66%), diabetes in 1 (11.11%). The precipitating event for TGA was emotional stress in 10 (55.55%), physical exertion in 2 (11.11%), hot bath in 2 (11.11%), not identified in 4 (22.22%). 4 patients (22.22%) were observed during the episode. 8 patients (44.4%)had reoccurring events. MRI disclosed thalamic lesion in 6 (33.33%), multiple lesion in 5 (27.77%) and was normal in 7 (38.88%).

Conclusion: These findings suggest that transient global amnesia may sometimes be associated with stroke.

Key words: transient global amnesia, stroke

BACKGROUND

Transient global amnesia (TGA) is defined as a selective memory impairment of acute onset and less than 24 hours duration. The pathophysiology of transient global amnesia (TGA) is not precisely clear; various brain regions may possibly be affected. TGA usually occurred in middle-aged or elderly people and was characterized by the abrupt onset of anterograde amnesia, accompanied by repetitive questioning. With the exception of the amnesia, there were no neurological deficits. The attacks lasted a few minutes or hours and the ability to lay down new memories was gradually recovered afterwards, leaving only a dense amnesic gap for the duration of the episode and of the hours leading up to it.

Clinically, it manifests with a paroxysmal, transient loss of memory function. Immediate recall ability is preserved, as is remote memory too; however, patients experience striking loss of memory for recent events and an impaired ability to retain new information. In some cases, the degree of retrograde memory loss is mild.

PATIENTS AND METHOD

Our series of patients with TGA were included in a prospective study during februar 1st2004- may 31st2009.

The diagnosis was based on positive clinical signs. Diagnostic criteria according to Caplan and Hodges or Hodges and Warlow, are: attack must be witnessed, acute onset of anterograde amnesia, no alteration in consciousness, no cognitive impairment other than amnesia, no loss of personal identity, no focal neurology or epileptic features, no recent history of head trauma or seizures, toxic substance intake were excluded. Attack must resolve within 24 h, and other causes of amnesia must be excluded.

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We recorded: risk factors, precipitating events, associated symptoms, duration, incidence in daytime and season, recurrence.

Laboratory tests, EEG, ECG and brain MRI were performed.

RESULTS

We observed 18 patients with TGA in the formerly mentioned interval of time. All patients characteristic are shown in the table 1 (IL = ischemic lesions).

Women were twice as numerous as men: 12 (66.66%) of them were women 6 (33.33%) patient were men. Age ranged between 36-72 years. Most of the patients were included in the 61-70-year-old group for both sexes. There was a definite difference between sexes as regards the age of occurrence: women were in their younger, men in their older age. The demographic characteristics of patients are shown in Diagram 1.

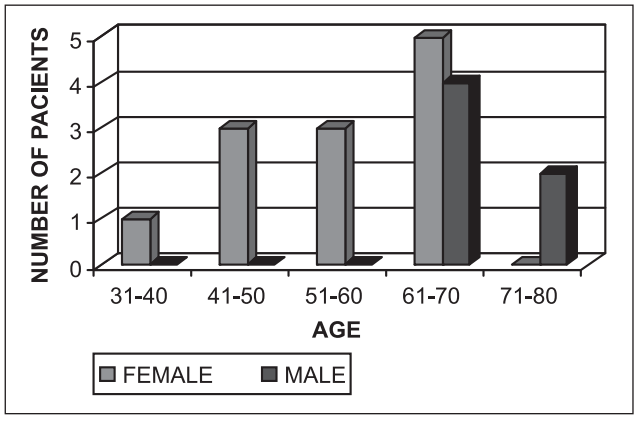


Diagram 1. *Demographic characteristic of patients*

The seasonal occurrence was the following: summer in 10 patients (55.55%), autumn in 6 patients (33.33%), spring in 2 patients (11.11%). No TGA episodes were recorded during winter time.

The daytime distribution was: evening in 7 (38.88%), morning in 6 (33.33%), and afternoon in 5 (27. 77%) patients.

We examined only 4 (2.22%) patients during a highly suggestive TGA attack, all of them strictly presented memory impairment and slight anxiety. The rest of 14 patients (77.77%) came in the moment when the attack had already been finished. All the patients presented an amnesic gap for the duration of the episode and none of them had persistent memory impairment afterwards.

The attacks lasted between 4 and 12 hours. The most frequent duration was 6 hours, found in 5 (27.77%) patients, 2 men (33% of men) and 3 women (25% of women) the duration of TGA was shorter in women. The time interval of TGA are shown in Diagram 2.

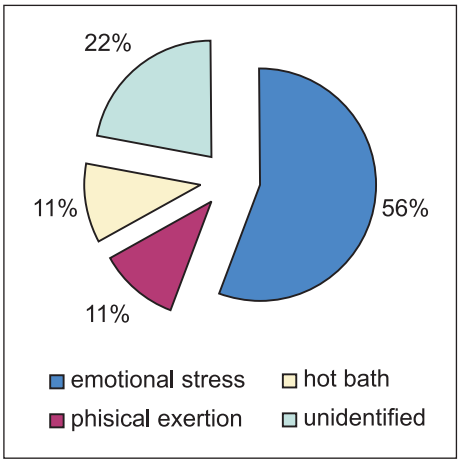


Diagram 3. *Precipitanting events for TGA*

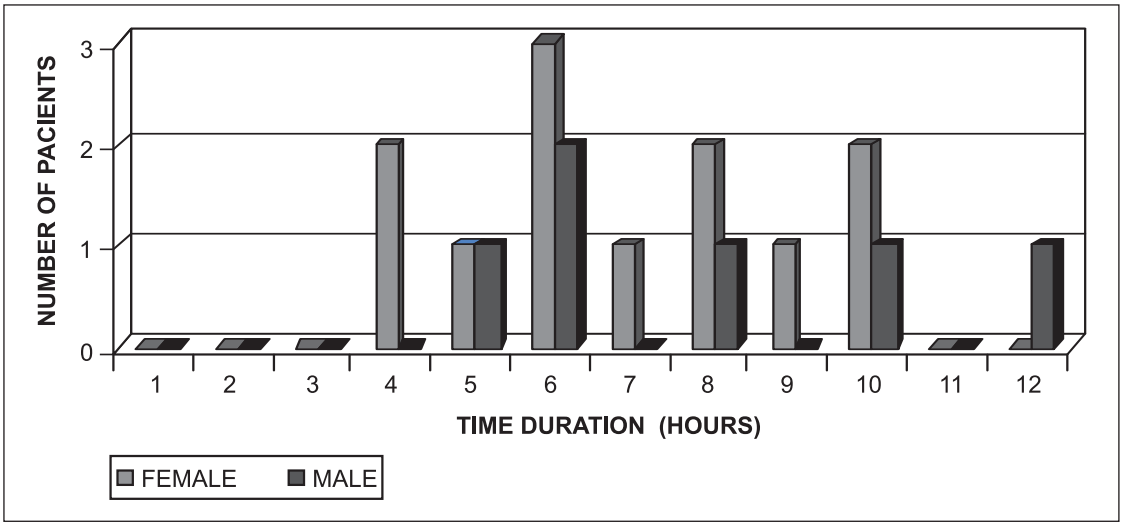


Diagram 2. *Time duration of TGA*

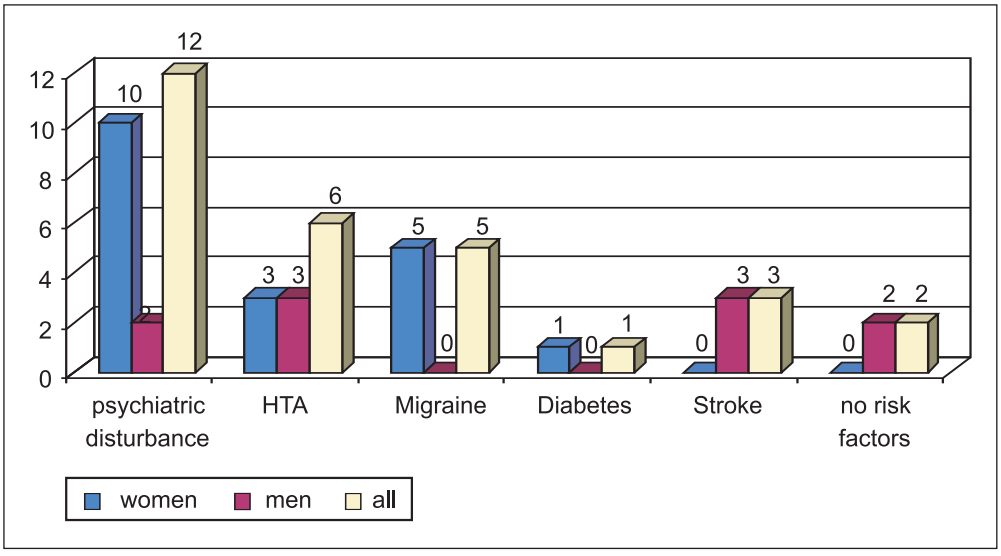


Diagram 4. *Risk factors for TGA*

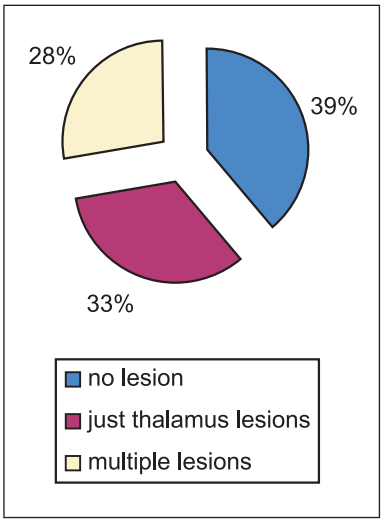


Diagram 5. *MRI*

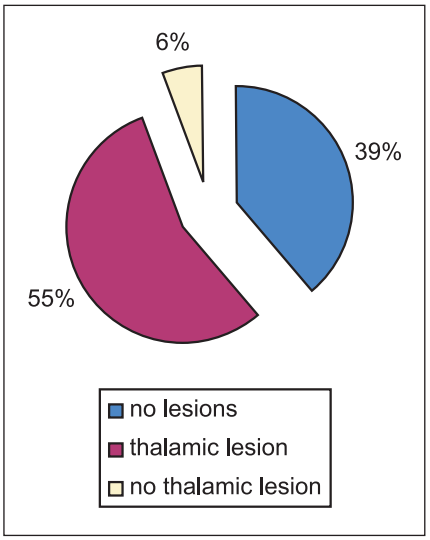


Diagram 6. *Thalamic lesions*

8 (44.44%) patients presented previous attacks of TGA, out of which 3 (16.66%) had recurring events. There was an 8 -24 months interval between episodes.

The precipitating event was emotional stress (death of a family member, conflictual situations) in 10 (55%), physical exertion in 2 (11,1%), hot bath in 2 (11.11%), unidentified in 4 (22.22%).

The following risk factors were identified: 1. Psychiatric disturbances (anxiety, depression, maniacal disposition) in 12 (66.66%) patients (10 women), namely 83. 33% of women and 33. 33% of men; 2. HBP was found in 6 (33.33%) patients (namely 25% of women and 50% of men); 3. Migraine was found in 5 (27.77%) patients, all women (41.66% of women); 4. Diabetes in 1 (5.55%), 1 woman; 5.3 (16.66%) patients, all men, had previous minor strokes, rapidly regressive in evolution.

In 2 (11.11%) patients of the 61-70 year age group, both men, we could not identify any risk factor.

Laboratory findings, EEG and EKG after the episode with unremarkable findings.

All the patients had a brain MRI at 7-10 days after the episode, which disclosed normal structure in 7 (38.88%), thalamic lesions in 6 (33.33%) patients (2 patients with bilateral lesions) and multiple lacunar infarction in 5 (27.77%). MRI findings are shown in images 1-11.

DISSCUTION

Our series included a relatively small number of patients with TGA. They were either admitted or treated in the out-patient clinic. Therefore, we could

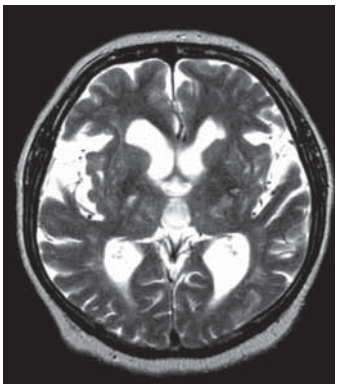


Image 1

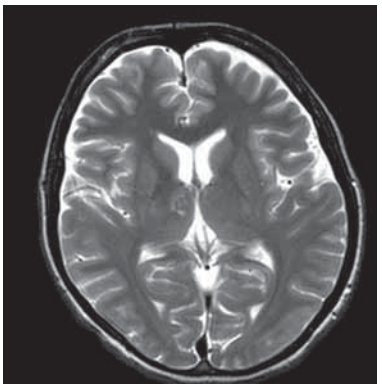


Image 2

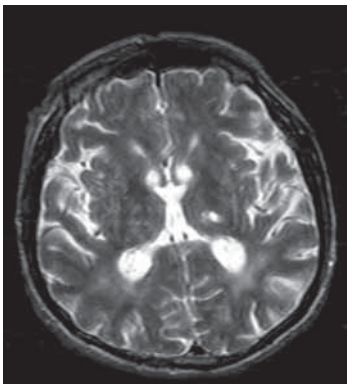


Image 3

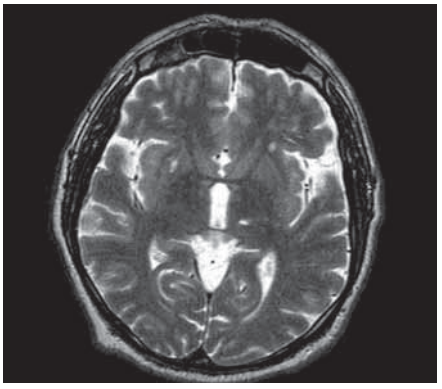
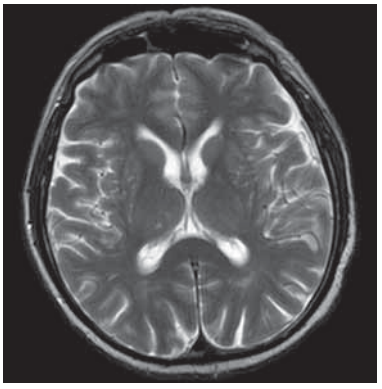


Image 4



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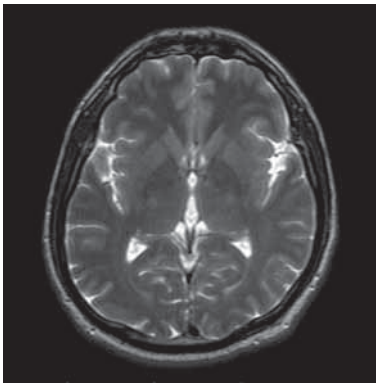

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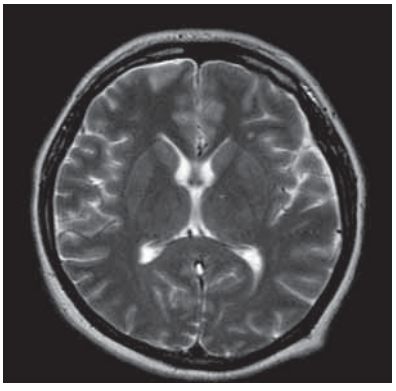

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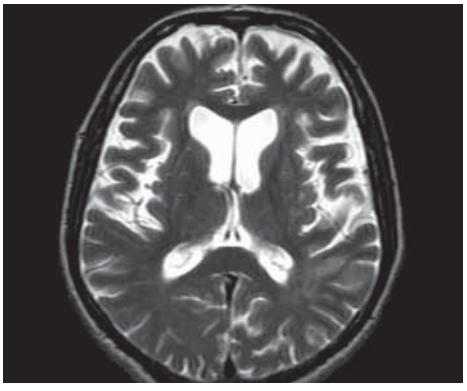

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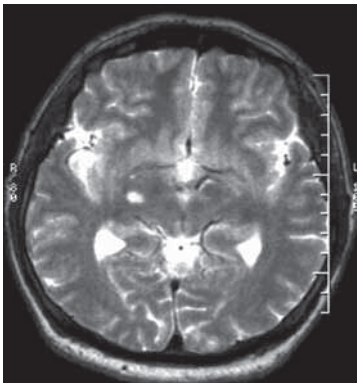

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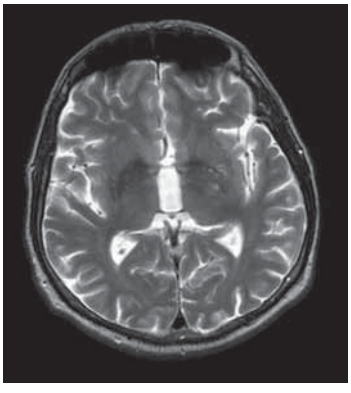

Image 10

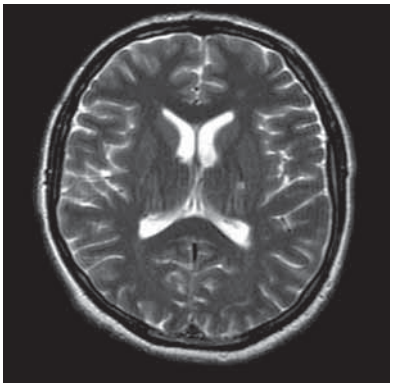

Image 11

not estimate the real incidence of TGA in our geographical area.

Women were twice as numerous as men, concordant with the female preponderance reported by the majority of authors (1-4). Only a few studies reported a greater incidence of TGA in men (5-6).

The age of the patients was placed between other reported limits (1-6), and the greatest number was encountered in the 61-70 years age group, for both sexes. Female gender definitely predominated in younger ages and male gender in older ages.

The duration of the attack, between 4 and 12 hours is concordant with other reports (1,6) and is placed in the middle range of the interval. We did not encounter extreme intervals. Women had a shorter duration of the attack in our series, especially the younger ones, occurrence that we did not find in other reports.

We found that emotional stress had a special value in patients that presented psychiatric disturbances. Physical exertion and hot bath seemed to be precipitating factors especially in older patients, with complex underlying disease. Our findings converged with other reports of multiple underlying mechanisms: neurotoxicity, ischemia, venous congestion. (3,7-9).

MRI disclosed a great proportion of patients with lacunars infarctions, although there were only 3 patients who had had stroke in their past history. 6 (33.33%) of patients presented with 1 one or several unilateral or bilateral thalamic lesions of various locations. Among the 5 patients with multiple lacunars infarction, 1 patient did not have any thalamic lesion, the rest had at least 1 thalamic lesion, and 2 had bilateral thalamic lesions. Several studies reported ischemic lesions in different brain regions during TGA (10-11), but we excluded cases with acute cerebral lesions, in order to fulfill diagnostic criteria. However, there have been reported cases with acute ischemic lesions in the context of a clinical picture of TGA (12-14). Similar clinical aspects, i. e. the abrupt onset of isolated memory disturbance were reported in different localizations of stroke (15-16). However, in these cases, the recovery was not that rapid and was not complete. On the other side, there also have been reported associations of TGA and other cerebral pathology, which could be accidental (17). In conclusion, it was interesting that only 7(38. 88%) patients had no brain lesions, and 10(55.55%) had poorly localisated thalamic lesions.

CONCLUSION

Although TGA is rather easy to diagnose, the clinical picture being highly suggestive, the indication for imagistic studies is strong, because these findings suggest that transient global amnesia may sometimes be associated with stroke.

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